son with normal controls disclosed no predominant blood group, HLA haplotype or qualitative difference in maternal-fetal incompatibility. These results suggested that an immune link must be associated with fetoplacental tissue-specific antigen. Although preeclampsia is a disease of primigravidae, often those multigravidae in whom this syndrome develops are pregnant from different fathers[8] and, therefore, I believe that it is the 50 percent of trophoblast contributed to by the father and its subsequent immunogenicity that sets the stage for this disease, rather than maternal immunological defects per se.

Typically one of three situations arises in a primigravida encountering antigen for the first time: The maternal antibody response may be poor in which case there is large antigen excess and no pathological immune complexes are formed. Second, those mothers in whom large antibody excess quickly develops become free of disease. However, third, in those mothers with an intermediate response, immune complexes are liable to be formed with slight antigen excess complexes that are extremely toxic and that will activate complement and aggregate leukocytes and platelets (apparently secondary to adhesion of the Fc fragment of antibodies to a specific platelet receptor) and damage endothelium producing a multisystem vasculitis.

In the past complement levels have been found to be no different between preeclamptic patients and normal.[9] However, this is a very crude test because to see a decrease in complement levels one would have to be activating complement at a rate that exceeded the ability to replace complement by increased synthesis or release. A more sensitive test is to look for byproducts of complement activation—C3a and C5a. Recent reports by Jacob[2,10] correlate the clinical severity of lupus with the degree of granulocyte aggregation and C5a levels in the circulation of patients. Current work (unpublished) is attempting to correlate the levels of C5a with the severity of preeclampsia-eclampsia. A number of interesting aspects are suggested by these findings: (1) The immunogenetic role of the father. (2) That superoxide dismutase and high doses of steroids (30 mg per kg of body weight of methylprednisolone)—drugs known to inhibit complement-induced leukoaggregation—may have a role in preeclampsia-eclampsia. (3) That abruptioplacentae may represent an exaggerated form of vasculitis, with increased tissue necrosis and consequent release of large amounts of thromboplastin and other antigenic material leading to a severe hemorrhagic diathesis usually not seen in the preeclampsia-eclampsia syndrome.

GRAHAM SIMPSON, MD
Las Vegas
Department of Medicine
University of Nevada, Reno
School of Medical Sciences

REFERENCES

1. Roberts JM: Preeclampsia and eclampsia—Medical Staff Conference, University of California, San Francisco. West J Med 135:34-43, Jul 1981

2. Jacob HS: Granulocyte-complement interaction—A beneficial antimicrobial mechanism that can cause disease. Arch Intern Med 138:461-463, Mar 1978

3. Petrucco OM, Thomson NM, Lawrence JR, et al: Immunofluorescent studies in renal biopsies in pre-eclampsia. Br Med J 1:473-476, Mar 1974

4. Arias F, Mancilla-Jimenez R: Hepatic fibrinogen deposits in pre-eclampsia. N Engl J Med 295:578-582, Sep 9, 1976

5. Kitzmiller JL, Benirschke K: Immunofluorescent study of placental bed vessels in pre-eclampsia of pregnancy. Am J Obstet Gynecol 115:248-251, Jan 15, 1973

6. Hulka JF: Antibody to trophoblast during early postpartum period in toxemic pregnancies. Am J Obstet Gynecol 86:130-133, 1963

7. Scott J: Immunogenetic factors in preeclampsia and eclampsia. JAMA 235:402-404, Jan 26, 1976

8. Need J: Pre-eclampsia in pregnancies by different fathers—Immunological studies. Br Med J 1:548-549, Mar 8, 1975

9. Kitzmiller J, Stoneburner L, Yelenosky PF, et al: Serum complement in normal pregnancy and pre-eclampsia. Am J Obstet Gynecol 117:312-315, Oct 1, 1973

10. Jacob HS: The scientific mind at work (and play). JAMA 246:25-29, Jul 3, 1981

Diagnosing Preeclampsia in Late Pregnancy

TO THE EDITOR: The Medical Staff Conference on preeclampsia and eclampsia[1] in the July issue was excellent. I felt it was a well-organized, well-presented, concise statement of the current knowledge.

I have been commonly faced with a situation not addressed in the article: A primigravida with normal blood pressure, no proteinuria and no edema evident on prenatal examinations enters the hospital in labor at term with a blood pressure of 140/90 mm of mercury, trace or 1+ proteinuria, minimal or no edema and hyperactive reflexes. The hypertension and hyperreflexia persist through labor. Is this patient preeclamptic? Does she need magnesium sulfate? If so, what dosage schedule?

DAVID G. STANGLAND, MD
Tonasket, Washington

REFERENCE

1. Roberts JM: Preeclampsia and eclampsia—Medical Staff Conference, University of California, San Francisco. West J Med 135:34-43, Jul 1981

* * *

Dr. Roberts Responds

TO THE EDITOR: The problem presented by Dr. Stangland is one that troubles a number of persons involved in the care of women during labor. This probably is one of the more striking examples